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# ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

## DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

## LEADING ARTICLES IN THIS NUMBER



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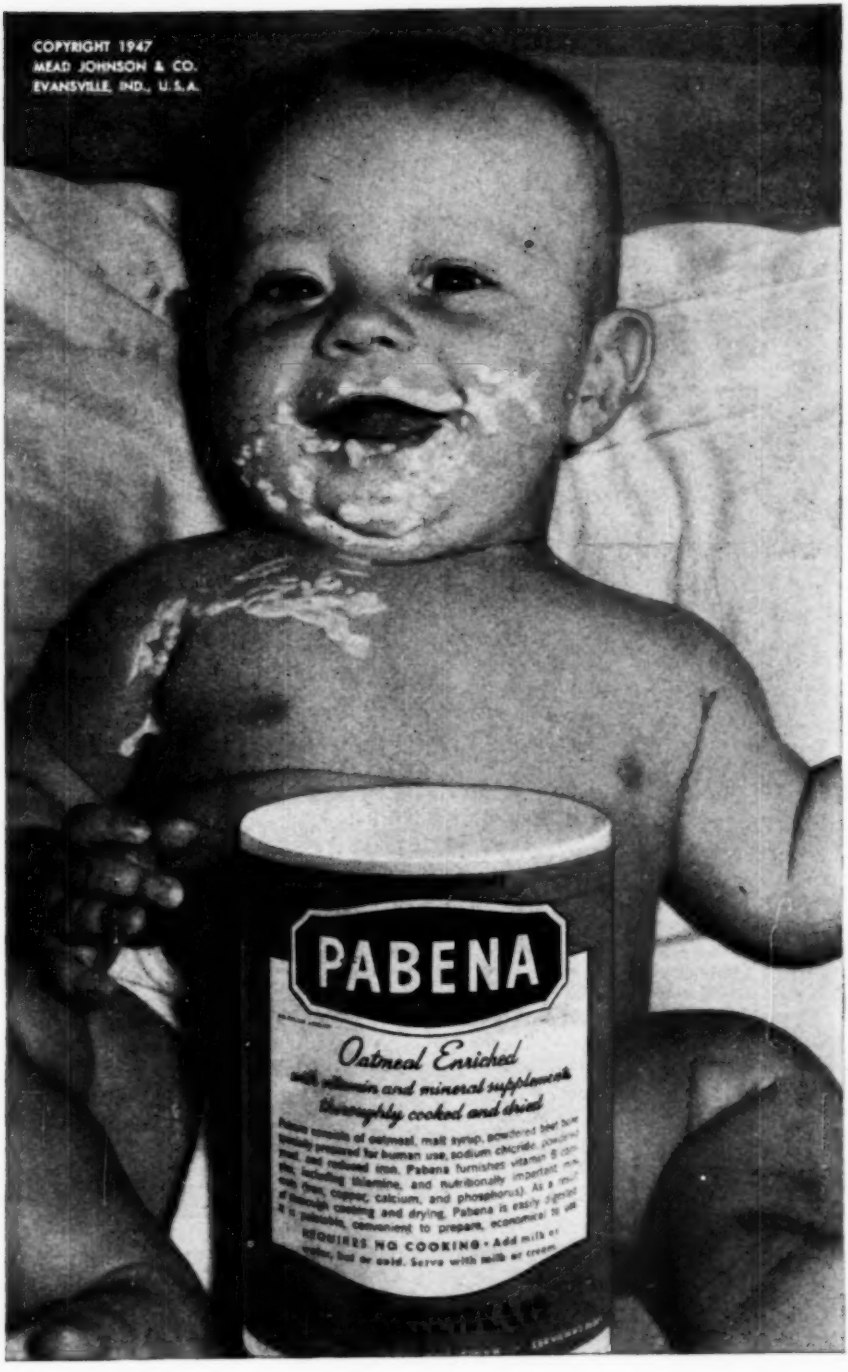
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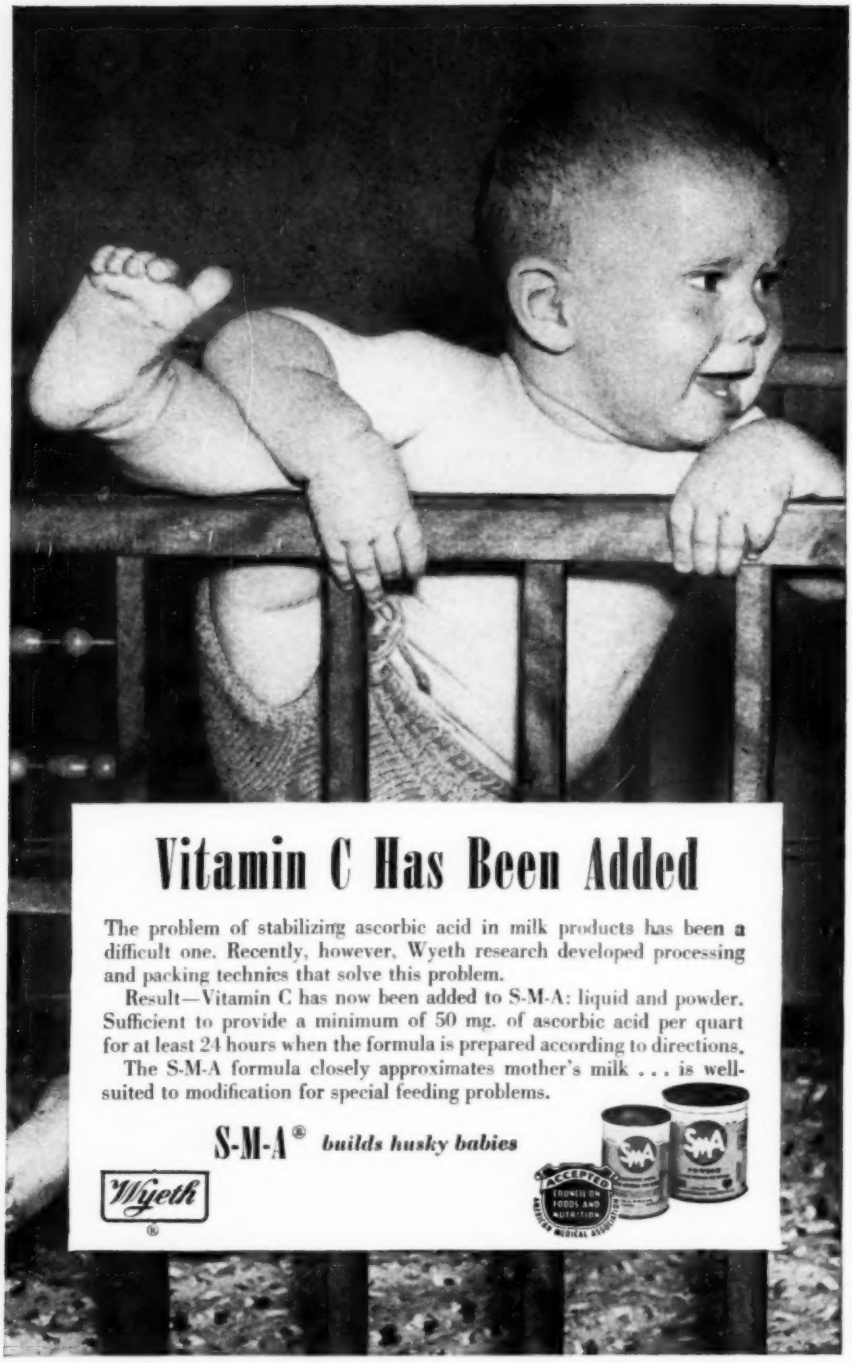

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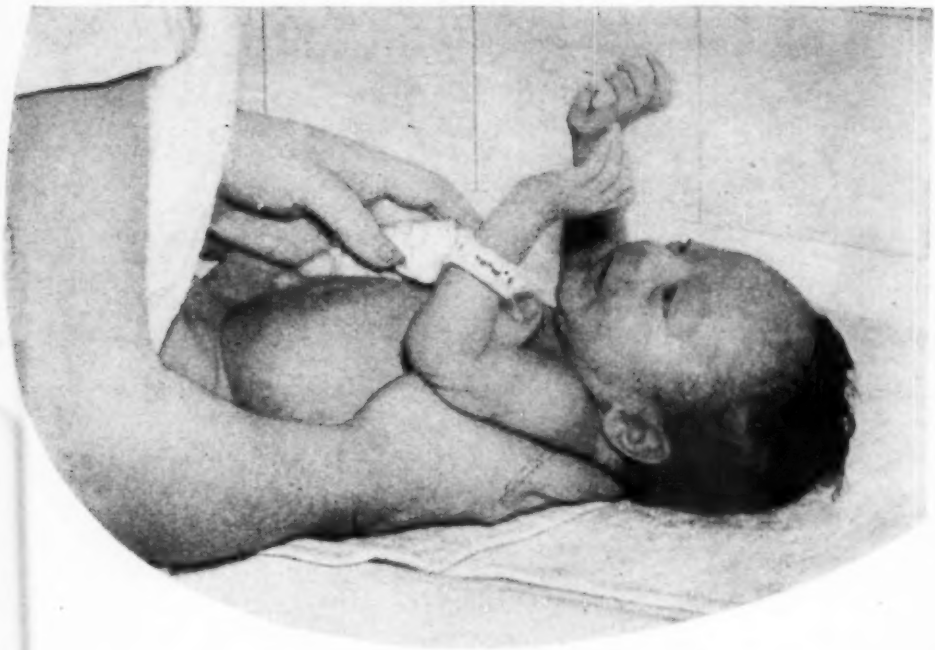

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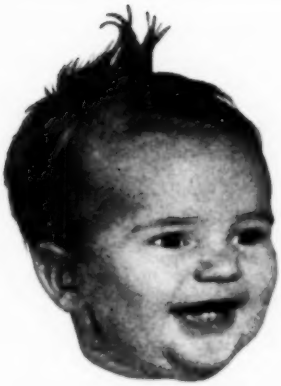

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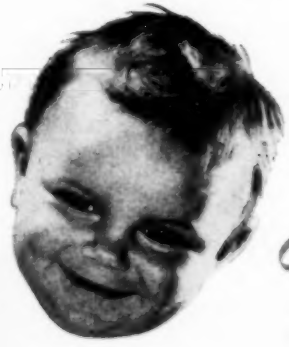

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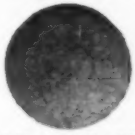
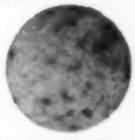


Film of Mennen Baby Oil (0.21 mm), 20,000 X. *Note homogeneous character.*



Film of evaporated baby lotion (0.21 mm), 20,000 X. *Note breaks, irregularity.*

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1. Elsner, H.—A Method for the Study of the Penetrability of Liquid and Semisolid Films Used in Skin Protection. Journal of Investigative Dermatology, Vol. 10, No. 4, April 1948. *Reprints upon request.*

2. Schwartz, L., Mason, H.S., and Albritton, H.R.—A Method for the Evaluation of Protective Ointments. Occupational Medicine 1:376-385 (April) 1946.

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(1) Follis, R. H.; Jackson, D.; Eliot, M. M., and Park, E. A.: Am. J. Dis. Child. 61:1 (July) 1941.

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*Frazer, J. G.: The Golden Bough, vol. 1, New York, Macmillan & Co., 1923

It is ironical that the practice of attempting to cure rickets by holding the child in the cleft of an ash tree was associated with the rising of the sun, the light of which we now know is in itself one of Nature's specifics.

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1. Flippin, H. F., and Reinhold, J. G.: Ann. Int. Med. 25:433, 1946.
2. Ledbetter, J. H., and Cronheim, G. E.: Am. J. Med. Sci. 216:27, 1948.
3. Frisk, A. R., et al: Brit. Med. J. 1:7, 1947.

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# ARCHIVES OF PEDIATRICS

VOL. 66 FEBRUARY 1949 No. 2

JOHN FITCH LANDON, M.D., Editor




## POLIOMYELITIS FOLLOWING TONSILLECTOMY

### A REVIEW OF THE LITERATURE


CORNELIUS H. NAU, M.D.

San Antonio, Texas.


In spite of the tabulation of many statistics showing the relationship of tonsillectomy to poliomyelitis, there have been few conclusive results derived therefrom. Review of the literature for the past six years shows several conflicting reports.

Among those stating that there is no relationship, Roberts,[1] who has collected statistics on cases of poliomyelitis reported in the literature for the 35 years prior to 1946, obtained figures that "show that the annual incidence of poliomyelitis in everyday life is 31 times greater than in the recently tonsillectomized—even during the poliomyelitis season."

Reporting a similar finding, Page[2] states that for the years 1937, 1939 and 1941, the Manhattan Eye, Ear and Throat Hospital sent out 27,849 questionnaires. Out of 8,915 replies, there was only one case of poliomyelitis reported. Cunning,[3] from the same hospital, states that in reviewing 11,204 tonsillectomy patients over a 7-year-period, only 4 cases of poliomyelitis developed after tonsillectomy, none being of the bulbar type. Cunning[4] also sent questionnaires to 13 states concerning 2,476 cases of poliomyelitis. Of this number he found that only 2.5 per cent had been tonsillectomized within the two months prior to the disease.

In a ten-year-period in Iowa (1937-1947) Kobayashi and Kehoe[5] found that only 0.8 per cent of the children had had tonsillo-

adenoidectomies done during a period of less than three months preceding the onset of their illness.

Hamilton,[6] of the University of Southern California, has found only questionable evidence of the relationship of tonsillectomy and adenoidectomy to poliomyelitis.

Winborn and Stansbury's[7] survey indicates that the incidence of poliomyelitis in tonsillectomized individuals is no greater than in those individuals whose tonsils have not been removed.

Reporting opposite findings, Anderson,[8] of Salt Lake City, quotes statistics on the Utah epidemic of 1943. He states that 43 per cent of the bulbar and bulbospinal cases were preceded by a tonsillectomy within 30 days of the onset of the disease. He found the incidence of poliomyelitis in recently tonsillectomized children to be 2.6 times greater than in the general child population, and the incidence of bulbar and bulbospinal types to be 16 times greater in the recently tonsillectomized than in the general child population.

Aycock[9] presented a comprehensive review of the subject in 1942, having collected data from the literature, personal communications and case records in Massachusetts (1927-31) and in Vermont (1912-31). His collected data show that the bulbar disease is five times greater than the spinal disease in those with recent tonsillectomy; that is, tonsillectomy within 30 days preceding the onset of the illness. His data also show a higher percentage of the bulbar disease in those individuals with a history of tonsillectomy (recent or more than 30 days preceding the illness) than in the non-tonsellectomized. His data cannot be analyzed as to whether or not tonsillectomy predisposes to clinical poliomyelitis. He questions the advisability of avoiding tonsillectomy because the chances of getting poliomyelitis are slight—much less than 1 per cent of the population get poliomyelitis, while 30 per cent of the population have had tonsillectomy.

Cary[10] records one case of bulbospinal poliomyelitis developing 15 days after tonsillo-adenoidectomy.

Perhaps one of the most convincing cases was that reported by Francis, Krill and Toomey[11] in which five children in one family developed bulbar poliomyelitis within 14 days after tonsillectomy, the unoperated child and both parents not developing the disease.

Hilding[12] has reported simultaneous poliomyelitis in four brothers, two of whom had been recently tonsillectomized. However, of three bulbar cases, only one had been tonsillectomized.

Toomey and Krill[13] also reported a larger series of cases showing the relationship. In a survey of 14 years of poliomyelitis at Children's Hospital in Akron and Division of Contagious Diseases, City Hospital, Cleveland, they found that "there were more individuals who had tonsillectomies and adenoidectomies within 30 days prior to onset of poliomyelitis, who developed a bulbar type of this disease than could be explained on the basis of mere chance or random sampling." Eighty-two per cent of those who had a tonsillectomy and adenoidectomy within 30 days prior to onset developed a bulbar disease; whereas, only 5 per cent of 140 non-operated patients admitted to Children's developed bulbar poliomyelitis, and 19 per cent of 118 admitted to City.

In reporting on the years 1937 through 1942, Lucchesi and La Boccetta[14] found that there were 432 poliomyelitis patients in the Philadelphia Hospital for Contagious Disease. Of 19 patients under 6 years of age that had had tonsillo-adenoidectomy, 41 per cent had bulbar poliomyelitis, while of 161 patients who had not had tonsillo-adenoidectomy, only 8.1 per cent had the bulbar disease. Seventy-eight per cent of 18 patients that died had had tonsillectomies and adenodectomies. The recentness of operation is not reported.

Seydell,[15] reporting on the Kansas epidemic of 1940, found that 46.6 per cent of those with the bulbar disease had had tonsillectomy, while only 14 per cent of those with the spinal type had undergone tonsillectomy.

Howard[16] made a survey of seven years (1937-1943) of poliomyelitis in Cincinnati. There were 233 cases occurring in July, August, September and October. Of this group only six cases developed following recent tonsillo-adenoidectomy. The ratio of poliomyelitis to tonsillo-adenoidectomy was 1:2000; the ratio of poliomyelitis following tonsillo-adenoidectomy to total cases of poliomyelitis was 1 : 40.

In reviewing 492 cases of poliomyelitis which were hospitalized in San Francisco during 1941-1945, Pedersen[17] found that the incidence of poliomyelitis following recent tonsillectomy is not greatly out of proportion to the ratio of the disease to the general population during an epidemic year; but that poliomyelitis, which occurs following tonsillectomy, is more apt to be bulbar in type. He found that there is a higher incidence of bulbar and bulbo-

spinal types of poliomyelitis in tonsillectomized patients than in the non-tonsillectomized, the ratio being 2 to 1.

There have been several explanations as to why bulbar poliomyelitis follows recent tonsillectomy. Cline[18] thinks that the "absence of the pharyngeal lymphoid tissue seems to be the factor rather than the recent traumatization to that area." Sabin[10] could not produce the disease in monkeys by applying the virus to the tonsillectomy wound; but after *injection* of the virus into the tonsillopharyngeal region, the disease did occur. This would indicate that in humans the bulbar disease occurs where the virus is already present at the time of operation, rather than there being contamination of the wound afterward.

## CONCLUSIONS

Conclusions which may be drawn at this time are:

1. Most reporters agree that tonsillectomy (recent or remote) does not predispose to poliomyelitis.

2. Many reporters have shown that if the disease does develop, the patient's chances of contracting the severe bulbar form are definitively increased if a recent tonsillectomy has been done.

3. In order to avoid the risk of bulbar poliomyelitis—although it is not great—operations on the nose and throat should not be done during epidemic months. However, during non-epidemic years the risk is minimal (one case in 2,000 operations[16]). The risk from November to June is nil except in Texas and California, where the poliomyelitis season is longer.

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DIPHTHERIA IN INFANTS. (Deutsche medizinische Wochenschrift, Stuttgart, 73: 389, Sept. 17, 1948). Goebel and Ströder point out that from the fall of 1945 till the summer of 1947 a decided change was noticed in the age distribution of cases of diphtheria. Whereas ordinarily those less than 1 year old accounted for only about 5 per cent of the total number of cases of diphtheria, during the aforementioned period the percentage was four times as high, and whereas formerly the mortality rate from diphtheria of the infants less than 1 year old had been less than 10 per cent, it was now nearly 36 per cent. A further analysis of the records of the infants with diphtheria revealed that the incidence and mortality were especially great during the first month of life. Of a total of 109 cases in infants under 1 year, 59 concerned infants in the first month of life and of these 26 were fatal, whereas of the 50 from the second to the twelfth month, inclusive, 7 were fatal. Discussing the possible causes of this increase in diphtheria morbidity and mortality in the youngest age groups, the authors say that only conjectures are possible. It has been suggested that inadequate passive immunization of the newborn by their inadequately nourished mothers may be a factor, because inadequate nutrition results in poor antitoxin formation. Some doubt is thrown on this supposition by the fact that the high incidence of diphtheria in young infants subsided again, while the nutrition of the mothers was still poor. The authors also discuss the possibility that the presence of other producers of toxins such as hemolytic streptococci, pneumococci and staphylococci, which were cultured from the organs of infants who had died, may have served as "pace makers" for the diphtheria toxin by increasing the permealibility of the blood-brain barrier and thus caused cerebral diphtheria.—*Journal A.M.A.*

# CELIAC SYNDROME DUE TO GASTRO-INTESTINAL ALLERGY

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Andersen[1], in a study of eighty-one cases, enumerates several causes of the celiac syndrome, but strongly minimizes food allergy as an etiological factor. She concludes that only two of her observed cases could be ascribed to gastro-intestinal allergy.

However, reports[2-5] have been appearing in the literature indicating that food allergy is a significant etiological factor for chronic and recurrent diarrhea in infancy and childhood. Lapin[2] states that in his experience colitis, due to a food allergy, is ten times as common as colitis due to a specific organism. To support his diagnosis, Lapin uses the sigmoidoscope. He describes the mucosa in allergic colitis as edematous, hyperemic with a diffuse blush.

Some authors[3,4] have made use of skin tests in an effort at reaching a correct diagnosis.

In the main, most clinicians rely on the history, the course of the disease and the dietary response.

It is the purpose of this paper to report some clinical observations in my cases of gastro-intestinal allergy, and to offer a simple laboratory procedure which I have found useful to differentiate gastro-intestinal allergy from other forms of celiac syndrome.

## LABORATORY TEST

It is a well-known fact that eosinophiles converge at the site of an allergic manifestation. The outpouring of these cells in allergic rhinitis is a constant observation. The stained smear of material from a vesicular hive will demonstrate a preponderance of eosinophil cells. Sputum from a child in an attack of allergic bronchial asthma is breath-taking with its concentration of eosinophiles. Likewise, the phenomenon of eosinophilia can be demonstrated in the mucus from the stool of a child with allergic colitis.

A particle of clear mucus is separated from the feces, spread on a glass slide, and stained with Wright's stain. The occurrence of eosinophilia is diagnostic of allergic colitis.

### SYMPTOMS

A recurrent or chronic diarrhea is the complaint that brings the patient to the office. A strict dietary regimen achieves stool improvement. Resumption of the normal diet precipitates a relapse.

The onset of symptoms varies from birth to eighteen months. This wide variation is determined by the introduction of an offending food, or the development of a specific food intolerance.

The following foods are usually implicated: cow's milk, banana, eggs, orange juice, apple sauce, fish-liver oil, chocolate, liver, cereals, vegetables. Cow's milk is the most frequent cause of symptoms.

The following specific food reactions are ascertained from the history:

1. Food dislikes.
2. Vomiting.
3. Sneezing, coughing.
4. Hives and eczema.
5. Colic, abdominal cramps and crying spells.
6. Loose or soft mucoid stools, frequency.

As an early symptom, a food may cause abdominal pain. Later, it may precipitate diarrhea. When multiple food sensitivity prevails, an attack subsides with the elimination of the offending food, but flares up when another intolerant food is added to the diet.

The patient is usually well developed and nourished. However, if the relapse is of long duration, irritability, poor appetite, sallow color and loss of vitality are evident.

### TREATMENT

Treatment of allergic colitis depends on the information gained from a thorough dietary history. All specific food reactions are ascertained. Foods for which there is any intolerance are isolated.

Treatment consists of the strict omission and elimination from the diet of suspect foods. A polyvitamin is added; and, occasionally, parenteral crude liver is administered.

Tolerance is achieved by this method of total avoidance of the indicted foods. In the experience of Rinkel[6], application of the rotatory diversified diet may also develop tolerance.

The readmission to the diet of an intolerant food depends on its

reactions. An early return is indicated when the food is the cause of a minor symptom. However, if it induces diarrhea, omission for one year is desirable. A fixed allergen is entirely discarded.

An early mild diarrhea, due to milk, frequently responds to the substitution for several months of boiled skimmed milk. A chronic, recurrent and severe diarhea caused by milk is affected only by the elimination from the diet of milk and milk-foods, and the substitution of non-allergenic milks, such as Nutramigen, Mull-Soy, goat's milk, boiled goat's milk and boiled evaporated milk.

## PROGNOSIS

It is my experience that tolerance develops successfully in twelve to eighteen months, and that the recurrence of sensitization is uncommon.

The causes for a relapse, while under treatment, are:

1. Respiratory infections.
2. Addition to the diet of unsuspected intolerant foods.
3. Resumption of offending foods before tolerance is safely established.
4. Development of a new sensitization.

## CONCLUSIONS

1. Food allergy is a significant etiological factor for chronic and recurrent diarrhea in infancy and childhood.
2. The phenomenon of eosinophilia can be demonstrated in the mucus from the stool of a child with allergic colitis.
3. The symptoms of gastro-intestinal allergy are precipitated by the introduction of an offending food, or the development of a specific food intolerance.
4. Treatment consists of the strict omission and elimination from the diet of suspect foods.

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# HISTORICAL

## BUDIN'S METHOD OF INFANT FEEDING

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There is a definite tendency at present, in prescribing a milk formula, to give infants more protein. Several articles in the recent literature illustrate this change in the practice of feeding. With the increase of protein, the carbohydrates in the food are usually reduced and the fat increased. Now we learn that so many babies receive evaporated milk—half canned milk and half water. In short, the baby is fed whole cow's milk without the addition of sugar. My own observations corroborate the conclusion of several pediatricians, that this formula provides a milk mixture that secures a satisfactory growth in most infants. I am not so sure about the absence of dyspeptic symptoms. In place of boiled cow's milk, evaporated milk diluted with an equal part of boiled water is preferred because it is sterile, homogenized and fortified with vitamin D. Vitamin C, of course, is a necessary addition.

The purpose of this article is to call attention to the studies of Professor Budin, more than fifty years ago. His pioneer work was published about 1900, and was translated by Dr. Walter Lester Carr, a superb volume of almost 200 pages,—and was printed by the Imperial Publishing Company of New York.

Pierre Budin was professor of obstetrics in the University of Paris and also medical director of a Consultation for Nurslings at the Charité in 1892; later he organized a similar service at the Maternité and also the Clinique Tarnier. Nowadays, we call such agencies "Feeding Clinics."

Sad to relate, the American edition of "The Nursling" had very little influence on pediatric practice in the United States. Pediatricians were then engrossed in the "percentage method" of infant feeding and the simplicity of Budin's method did not appeal to us.

Today it will pay everyone, who treats infants, to study this book (if it is still in print). The American edition was printed in 1906. At that time I took charge of premature infants in a St. Louis institution and the precise directions that Budin gave

for the care of the premature infant aroused my admiration and I have followed them ever since.

However, here we are concerned only with the artificial feeding of the normal infant. Let me quote:

"Should cow's milk be given undiluted? It is generally supposed to be harmful to infants, owing to the excess of casein it contains compared with the human product. To counteract this defect great quantities of water are usually added to the milk. The dilution varies with age; at first, three parts are added to one of milk, then two; then equal parts of each are used, then two milk and one of water, and so on to the fifth or sixth month, when undiluted milk is reached. But casein is not the sole constituent of milk. It contains also butter, sugar and salts, all of which contribute to its value as food. Diluted cow's milk is generally deficient in these substances, and therefore forms but a poor substitute for a mother's milk. To obtain sufficient nourishment on this alternated diet, infants are obliged to absorb great quantities of fluid, which causes them to pass great quantities of urine. They are always crying from hunger, whereas infants on undiluted milk wait contentedly for their next meal.

"It is alleged that the casein of cow's milk forms large clots in the stomach and gives rise to digestive troubles. This is obviated if the milk is heated in a sterilizer at a temperature of 100° C."

Then he quotes the investigation of Chavane to support his contention. "Sterilization is greatly simplified by the use of undiluted milk."

"When I first advocated the feeding of infants on undiluted milk, critics asserted that infants could neither support nor digest it, and that it was responsible for quite a catalogue of evils—digestive troubles, rickets, eczema, urticaria, etc. I have never seen any bad trouble from its use, and infants thriving on it may be seen daily at the Consulation. The milk I distribute contains 37.35 grams of butter, on the average, per liter.

"I carefully refrain from asserting that during the first few months of life milk ought invariably to be prescribed unmixed with water."

Even mother's milk when it is too rich may not be tolerated. "Cow's milk is not of constant composition—it varies with the breed of animal, the feeding and other factors."

Sick infants require that their digestive tube has need of judicious treatment. "It may be of service to add a variable quantity of ordinary water, barley water, lime water or other diluent to the milk."

"It is more than probable that the evil effects attributed to rearing young babies on undiluted milk are in reality due to over-feeding."

Budin refers to several French pediatricians who have become partisans of undiluted milk and he cites Variot, Comby, Lazard, Drapier, Ruffie, Bonafas and several others.

How much undiluted sterilized milk ought a baby receive? Budin answers this question extensively and reports many interesting cases from his practice. He rejected the capacity of the stomach as a guide, as it is too difficult to estimate. The age also is an unreliable standard, even the weight is a poor guide.

"Infants who are underfed are free from digestive troubles. They do not gain in weight, but, by gradually increasing the quantity, it is easy to attain in safety an adequate diet for a given case, and then the child will grow rapidly." Budin insists on human milk for all young babies, but "if mixed or artificial feeding be a necessity, there are three cardinal laws which govern the use of undiluted milk. First give milk of good quality. Second give milk in correct quantities, neither too much nor too little. Third sterilize the milk."

Weighing the baby regularly gives Budin's main index for adjusting the quantity of milk. If no gain in weight occurs and no other cause is discovered, the baby's diet should be increased.

At that time the value of estimating the caloric value of the food had not been discovered, consequently, Budin had to depend almost entirely on the reaction of the infant. Give a small quantity and gradually increase this until the baby gains in weight and is comfortable.

He reports a number of cases of indigestion due to over-feeding. The supervision of the feeding is necessary in artificial feeding and special attention should be paid to the quantity.

In order to accentuate the truth of his personal impressions he narrates the following: "A distinguished doctor came from Budapest not long ago in order to see my Consultation. He had been accustomed to believe all sorts of accusations against undiluted

sterilized milk as an infant's food. To convert him from error and remove his doubts, I had all the children stripped, and then invited him to examine them. This he did with the greatest care, but to his profound surprise did not find a single child which presented even a trace of rickets."

Budin claimed that his infants are not subject to large bellies, that their muscles are not flabby and have very good resistance to disease. He is unacquainted with the so-called "undiluted-milk dyspepsia."

Not a single case of rickets had occurred among the children for three years or more. "Neither will you see eczema among my patients." "As for the so-called 'infantile scurvy,' which is alleged to follow the use of sterilized milk, I have heard a great deal about it during the last few years, but I am still looking for my first case."

The selected quotations from Budin's book clearly represents Budin's method. Little was written about how he sterilized his milk. The older pediatricians will recall the use of the Soxhlet's apparatus for sterilizing milk. Several improved utensils for the sterilization of milk were offered at that period. In fact, Budin described his own apparatus, but he admits that boiling the milk is just as efficacious.

As already stated, American pediatricians did not subscribe to this formula, undiluted cow's milk, although it was prescribed in some of the feeding clinics which were organized in the large cities of the United States about that time.

What now? Shall we generally subscribe to this formula? Undiluted, sterilized cow's milk—or its safer imitation—evaporated milk and an equal quantity of sterile water?

In my own practice I have avoided a milk formula in which the protein-nonprotein ratio (nutritive ratio) is more than ⅕ or 1/6. This corresponds to ⅓ evaporated milk, ⅔ water with 4 or 5 per cent of carbohydrates. Before adopting the "undiluted milk" formula we need to know several facts, and these are hard to procure. Here are the questions:

**1.** Does undiluted cow's milk favor the early closure of the fontanel and sutures? I think it does. (See ARCH. PEDIAT., 61: 250-255, May 1944).

**2.** Does undiluted cow's milk favor the ammoniacal diaper? It certainly does.

3. Is there a difference in the specific dynamic action of protein of whole cow's milk and diluted cow's milk?

4. As the infant obtains so much of its energy from protein, does this train the digestion to tolerate carbohydrates? If this is true it can be easily overcome by feeding cereals early.

5. Does this over-feeding with protein hasten maturity and shorten the period of childhood?

6. Have several hundred children been studied, one-half whole milk and one-half on a diluted milk? It has not been done.

7. What shall we say about several proprietary foods, the composition of which show a low protein content, yet are widely prescribed?

8. What about the vitamin content?

This is sufficient. Anyone may ask questions. One can not resist the impression that the formulae of infant feeding at present are so many and variable in composition that in reality there is no science of infant feeding. Some of our younger men should clear up some of these problems.

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PENICILLIN IN GONORRHEA IN CHILDREN. (Archives Argentinos de Pediatria, Buenos Aires, 19: 335, June 1948). Wiederhold and Méndez administered penicillin to two groups of infants and children, of 25 children each, with gonorrhea. Most of the children were girls. Children in the first group received penicillin by mouth in a total dose which varied from 200,000 to 1,500,000 units, the children in the second group receiving the drug by intramuscular injection in oil in one dose which varied from 200,000 to 250,000 units. The patients were observed for two years after clinical and bacteriologic recovery. Tests for reactivation of the disease were performed during this period. Permanent clinical and bacteriologic cure was obtained in all patients who received penicillin by mouth. Permanent clinical and bacteriologic cure was also obtained in 20 of 25 patients who had injections and in 5 patients in this group who had penicillin by mouth after failure of penicillin injections. The authors believe that administration of penicillin by mouth is more practical, is painless and more efficient than penicillin by injections.—*Journal A.M.A.*

# CLINICAL REVIEWS

*In order to encourage the writing of clinical articles by recent graduates or senior medical students, the* ARCHIVES *will publish monthly at least one such paper from the classes of Doctor Reuel A. Benson, New York Medical College, New York; Doctor Philip Moen Stimson, Cornell Medical School, New York, and Doctor John A. Toomey, Western Reserve University Medical School, Cleveland. Other interested medical schools are cordially invited to submit student papers for consideration.*

## INTERVENTRICULAR SEPTAL DEFECT WITH STOKES-ADAMS SYNDROME*

REPORT OF CASE

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The interventricular septal defect, or maladie de Roger, is one of the most common congenital deformities of the heart. Abbott[1] has found in the course of 1,000 autopsies on those with congenital heart disease that 257 had an interventricular septal defect. This case is reported because of its unusual involvement of the bundle of His, giving rise to a Stokes-Adams syndrome. This case is especially interesting since its diagnosis may be made on the basis of the history and the clinical examination without the elaborate angiocardiography and cardiac catheterization, which may be useful in the diagnosis of the other congenital heart lesions.

### CASE REPORT

The patient was a seven-year-old Puerto Rican male, admitted to the Metropolitan Hospital, New York City, on December 29, 1947. He was brought to the hospital because of an upper respiratory complaint; however, in the course of history taking, a more serious condition was immediately suspected.

The child's delivery was a normal one, and his development seemed to follow normal limits, but his growth was retarded. Two years prior to this admission, in Puerto Rico he began to have attacks characterized by a cry, tenseness of all muscles with partial loss of consciousness. He never fell to the floor, had convulsions, or bit his tongue. During the course of an attack, he

*Submitted as partial fulfillment of the requirements of the course in Senior Pediatrics at the New York Medical College, Flower and Fifth Avenue Hospitals.

was said to become very cyanotic with copious, frothy sputum issuing from the mouth. These attacks would last from fifteen to twenty minutes. For the following year and one-half he had the attacks frequently—two or three times a week. There were days when he would have as many as three or four attacks a day. The attacks seemed to be brought on by emotional excitement, punishment, etc.

The patient has six siblings all of whom are living and in good health. Careful questioning revealed no suggestion of epilepsy in the family. The rest of the family history was noncontributory.

The patient was free of attacks for a three-month period, but, on traveling to the United States by airplane, three months before admission, he was seized with an attack. He has had three attacks during the past three months that he has been in New York City. He was receiving pills in Puerto Rico for these attacks which his parents believed prevented some of the attacks. The child has been dyspneic on slight exertion since birth. Cyanosis was apparently not present at birth. He now has cyanosis only during the course of an attack. There had been no real orthopnea, palpitation, ankle edema, chest pain, paroxysmal nocturnal dyspnea, or any of the other signs of congestive heart failure. Questioning with regard to joint pains, nose bleeds, abdominal pains and other significant rheumatic fever symptomatology was noncontributory. Careful review of the symptoms by systems gave no pertinent information; those positive answers being insignificant.

The child's health had generally been poor with frequent upper respiratory infections and one bout of pneumonia. He had the pneumonia two years ago. The child's eating habits were poor and consisted of the national Puerto Rican diet—beans and rice. His emotional development on cursory observation seemed to be adequate though his education had been neglected.

Physical examination revealed an obviously underweight seven-year-old boy. He weighed 38 pounds and was 43 inches tall. He was not cyanotic, though his lips were dusky in color. The tonsils were hypertrophic, cryptic and injected. The pharynx was also injected. The right tympanic membrane showed superficial injection. There was lymphadenopathy involving the anterior and posterior cervical, the posterior auricular and inguinal glands. The glands were large, discrete and painless.

There was a definite cardiac thrust, the point of maximal impulse being in the fifth intercostal space, one centimeter to the left of the midclavicular line. The left border of the heart was percussed about one centimeter beyond the midclavicular line in its most lateral extension. The right border of the heart did not seem enlarged. The heart was not mitralized to percussion. On auscultation the rhythm of the heart was regular—48 per minute. A prolonged, loud, blowing systolic murmur followed by a short rough diastolic murmur was heard over the entire precordium but loudest over the fourth intercostal space just to the left of the sternum. Over the same area a systolic thrill was palpated. The blood pressure was 104/60.

The lungs showed bilateral dullness at the bases with occasional râles and some expiratory ronchi; respirations were 28 per minute.

*Laboratory Work.* Urinalysis: Specific gravity 1,020; there were no red cells, white cells, casts, albumin or glucose in the urine.

Blood count: Hemoglobin 11 grams. R.B.C. 3,200,000. W.B.C. 11,500—polys 82 per cent, lymphs 18 per cent.

Sedimentation rate: 14 mm. at the end of an hour.

X-ray: "Increase in the transverse diameter of the heart with marked rounding of the left ventricular border. Concavity in the region of the pulmonary conus."

Electrocardiogram: Complete heart block; bundle branch block —common type.

The child's course in the hospital was not eventful. He was treated principally by diet, which was high in calories, proteins and vitamins. 0.5 per cent neosynephrine, two drops in each nostril, was also used. The upper respiratory infection yielded within a few days to this therapy. After about two weeks of hospitalization, the child was discharged to the cardiac clinic. He had experienced one attack of cyanosis while he was in the hospital.

## DISCUSSION

The interventricular septal defect was first described by Roger in 1879[2] and from then on the condition came to be known as the maladie de Roger. The case under discussion is one of the few cases in which the lesion lies in the lower part of the septum and was first adequately described by Weiss.[3] Localized defects of

the interventricular septum are usually situated at the base of the heart just anterior to the pars membrancea and open on the side of the right ventricle. The defect is usually small—the intensity of the murmur varies inversely with the size of the defect. The character of the murmur in this case suggests a small opening which unfortunately is low in the septum giving rise to the Stokes-Adams syndrome which will be discussed later. Ordinarily, without involvement of the conduction system, cyanosis is absent except as a transient or terminal feature and is even then rare. The clinical picture is that of absence of symptoms in the presence of very distinctive physical signs in an otherwise normal individual.

The etiology of congenital heart disease is obscure and the case under discussion is no exception. Dogaramaci and Green[4] investigated 434 patients with congenital heart disease and found that mumps, rubella and scarlet fever during pregnancy certainly do not constantly produce offspring with anomalies. Though a small percentage of cases may be caused by the above mentioned diseases during pregnancy, as well as exposure to noxious agents, the vast majority of cases remain unexplained. The importance of heredity as a factor is undoubtedly great, but this is obscured by the propensity of such defects to cause early death of the fetus. The authors noted with interest that in 9.5 per cent of the cases parental lead exposure was noted.

In the interventricular septal defect a shunt exists so that blood flows from the left ventricle to the right. The pressure in the left ventricle is normally greater than that in the right. Hence, there is no cyanosis until for some reason the pressure in the right ventricle becomes greater than that in the left, or there is involvement of the bundle of His. When venous blood flows from the right ventricle to the left, cyanosis comes on. The exact rate in complete heart block depends on the site of the idioventricular pacemaker. When the lesion is higher in the a-v node, the rate will be faster. Danger arises when the ventricles stop contracting entirely. Such pauses come suddenly and the symptoms they produce will depend on their length. If the pause lasts several seconds, the patients may only feel a faint wave or light-headed sensation, like a petit mal. If it lasts a little longer, they faint away for several seconds. If it continues for twenty to sixty seconds, they will lose consciousness and have a convulsion, the for-

mer being the situation with our case. If it lasts a few minutes, breathing ceases and death generally results. The bundle of His is rarely involved as in this case and there may be disturbance of rhythm from delayed conduction, partial a-v dissociation, to complete heart block. The recognition of the condition at the bedside has been outlined as follows[5]. 1. Partial heart block is recognized by a third sound in diastole. 2. Incomplete by a regular rhythm with true dropped ventricular beats. 3. Complete heart block by an irregularly occurring third heart sound in diastole and occasionally accentuation of the first heart sounds caused by the superposition of an auricular beat on a ventricular beat with a complete dissociation.

The distinctive, long, loud, harsh systolic murmur best heard over the fourth intercostal space just to the left of the sternum accompanied by a thrill over the corresponding area all but makes the diagnosis. The murmur is quite characteristic and in a series of 12 infants and children whom Gibson[6] followed to autopsy in whom a defect of the interventricular septum was found, the typical systolic murmur described had been noted in 11 cases. In the remaining one, the infant was moribund on admission to the hospital and the physical examination was not completed. Therefore, it may be said that the diagnosis on auscultation may be made with a good deal of assurance when it exists in pure form.

In this case the electrocardiogram is unnecessary in the making of the diagnosis, for the obvious defect in the conduction system is made evident by the physical signs and history of syncope. However, the electrocardiogram gives us an objective record which tells us that, in this case, we are definitely dealing with a complete heart block; this of course may be done clinically as was outlined above. The electrocardiogram would seem to be helpful in the diagnosis of a defect in the interventricular septum by the absence of marked right ventricular preponderance, by the presence of no axis deviation, or perhaps slight right preponderance; by the absence of biphasic Q.R.S. complexes and regular sinus rhythm; and, when present, the manifestations of conduction disturbances, as in our case, help to establish the diagnosis.[7]

Although the x-day was not needed in making the diagnosis of this case, any case of congenital heart disease today demands dis-

cussion of its use. In recent years, two methods that are helpful in certain cases of congenital heart lesions have been added to the diagnostic armamentarium. They are catheterization of the heart and angiocardiography. Sosman and Dexter[8] have given an excellent description of the indications, technic, errors, results, interpretation and value of heart catheterization. The pressure in these various parts of the heart and large vessels is determined through the catheter, and blood samples are obtained for oxygen-content determination. At the time of publication, the authors had performed catheterizations in 100 cases without mishap. Angiography consists of radiography of the heart and great vessels following rapid injection of 30 cc. of 70 per·cent Diodrast.[9] The position, size and shape of the heart chambers, valves and great vessels have been studied, and knowledge obtained that had not previously been available. The brilliant developments of surgery of certain of the other congenital anomalies have made it imperative that an exact preoperative diagnosis be made.

Ordinarily a defect of the interventricular septum does not impose any appreciable strain on the heart; however, in nearly 40 per cent acute bacterial endocarditis is said to develop.[10] This is in direct contrast to the analogous interatrial defect which has not been described as a site for subacute bacterial endocarditis. The patients usually succumb to subacute bacterial endocarditis, tuberculosis and other terminal infections. Some of the cases die of acute heart failure. Abbott,[1] in her 257 cases, found the average age of death to be 14½ years, the highest being 49 years. Some patients with complete heart block may have a favorable prognosis regarding life and the ability to perform daily tasks. Campbell[11] states that if there are no complications carrying special risks of their own, the prognosis is good and will probably prove that the condition is compatible with survival to old age. Only a small percentage of those with congenital heart block experience Stokes-Adams attacks.[12] In a series of 8 cases that Campbell[11] followed for nine years, he reported 2 deaths. The average survival rate for the other 6 patients was 22 years on last examination. With this encouraging report of prognosis in congenital complete heart block and others by Jaleski and Morrison[13] (31-year-old woman who went through two terms of pregnancy and delivery without complications) and Levine[5] (mentions a 55-year-old patient who

was known to have had complete heart block since the age of six), we must still feel that the prognosis in the case reported should definitely be guarded, because of the frequency of syncopal attacks. The precarious feature of complete heart block is not the slow regular rate of 48 but the temporary complete failure of the idioventricular pacemaker to send out impulses. Our patient seems to have a strong tendency to do this with the resultant syncopal attack which may at some time end his life.

The therapy in this case is twofold, and in each instance the prophylactic aspect is probably the most important. The patient should be followed about every two weeks in the cardiac clinic and careful observation be made for any signs of incipient subacute bacterial endocarditis. With the early diagnosis of subacute bacterial endocarditis and its early treatment with adequate dosage of penicillin, the child may be freed from the previously great danger of this complication.

Treatment is not directed at the slow steady rate, for this is productive of no symptoms. However, the main concern is the prevention of the attacks of syncope and their treatment once they occur. Ephedrine sulfate (0.025 gram), two or three times a day, may prove effective. In stubborn cases a great variety of procedures have been employed in treatment. Amongst these are inhalations of 1:1000 adrenalin, benzedrine, propadrine, full doses of atropine intravenous injections of 50 per cent glucose, thyroid extract, cardiazol and digitalis. When attacks occur frequently, many a day, as may happen, adrenalin given subcutaneously, may be lifesaving.

## SUMMARY

A case of interventricular septal defect with complete heart block is reported. The anatomy, etiology, pathologic physiology, diagnosis, prognosis and treatment are extensively discussed.

The case is presented as one in which the clinical diagnosis is adequate without resorting to the more elaborate diagnostic methods recently made available for the diagnosis of other abnormalities.

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PENICILLIN IN HEMATOGENOUS OSTEOMYELITIS. (Acta Chirurgica Scandinavica, Stockholm, 96: 547, June 28, 1948). The material reviewed by Vuori and Sulamaa was collected from several hospitals. It comprises 83 cases in which 92 bones were involved. Ten patients were adults and the remainder children below 17 years. The infecting organism was determined in 60 patients; in 2 streptococci were found; in 1 both streptococci and straphylococci; in 1 Staphylococcus albus and in the remainder Straphylococcus aureus. The treatment varied considerably in the different hospitals. Treatment with penicillin was often commenced rather late. One patient died probably because of insufficient dosage of penicillin. In the majority of cases the effect of penicillin on the general infection at the initial stage was good. The disease became chronic in spite of the treatment with penicillin in 42 cases, and did not heal until sequestrectomy or radical trepanation had been performed. The treatment with penicillin gave better results in the cases of primarily chronic disease than in those of acute disease; it was more effective in the cases which showed principally osteoplastic roentgenologic bone changes than in those showing principally osteolytic changes. No significant correlation could be proved between the recovery and the intensity of the treatment with penicillin and the time of its commencement. The fact that the group of cases in which treatment with penicillin was given within the first week of illness contained a significantly larger number of cases of the osteoplastic type than the group which came under treatment later indicated that it is important to aim at early treatment. Patients treated with smaller doses had a lower frequency of complications.—*Journal A.M.A.*

# REVIEW OF THE LITERATURE

## THE EPIDEMIOLOGY OF POLIOMYETITIS*

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*(Continued from the January issue)*

The development of specific humoral antibodies produced by exposure is further rendered questionable by studies on humans. In one small series of 14 acute cases, there was no demonstrable increase in the circulating antibody titre three months after the acute stage. Nor did contacts who lacked virucidal antibodies at the time of exposure, develop them subsequently.[36] This work has been questioned on the grounds that the virus used to detect the presence of antibodies was a stock strain and not a freshly isolated strain. But, concurrent studies confirmed the conclusion that active infection had failed to produce a significant change in antibody titre and there was no difference in the results obtained whether the antigen used was freshly isolated or stock laboratory strain of virus.[36] This problem might be resolved by immunological studies of the sera of acute and convalescent cases, using virus isolated from each patient as the antigen. This would rule out error due to mistaken diagnosis as well as the error that might result if more than one strain of virus were implicated in the epidemic. In any event, these results have led the investigators to the conclusion that virucidal antibodies and immunity do not necessarily result from contact with the virus, but rather develop as part of the process of attaining maturity.[38] There is evidence, however, that specific tissue immunity does develop after infection. In animal experiments, it has been found that a monkey, which has survived a paralytic attack, is not susceptible to intracerebral re-inoculation with the same strain of virus. This resistance was independent of the presence of virucidal agents in the circulating blood.[35] This is substantiated by the observation that a frank attack of polimyelitis usually produces long lasting immunity in humans, although second attacks do occur.[3]

It is highly probable that the most important factors which

*In September 1948 this essay was awarded first prize by Philip Moen Stimson, M.D., in a competition open to his third year students at the Cornell University Medical College for theses on a subject in the general field of contagion in children. Mr. Davolos will receive his M.D. in June 1949.

determine the susceptibility or resistance of individuals, who have never had clinical poliomyelitis, are nonspecific.

Age of the Patient: Age plays an important part in the susceptibility to the disease and the severity of the symptoms which result. Poliomyelitis is primarily a disease of childhood, most of the cases occurring between the ages of one and fifteen. But, when the disease does occur in older individuals, it is usually more severe and carries a higher fatality rate. The contrast between the age-distribution curve and the age-specific lethality curve is depicted graphically in Fig. 4.

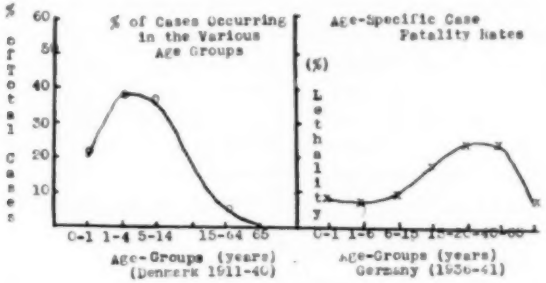



Fig. 4. These graphs are designed to contrast the age distribution of poliomyelitis with the case fatality rates in comparable age groups (constructed from data in Bertinius,[39] Acta. path. et microbiol. Scandinav., Supp. LXVIII, 1947).

Influence of Fatigue, Chilling and Trauma: The frequent history of violent exercise preceding the onset of symptoms of acute poliomyelitis has prompted one group of investigators to study the predisposing effects of certain physical factors. Their findings revealed that monkeys, forced to undertake exhausting exercise during the incubation period, had a significantly higher incidence and severity of the disease than well-rested controls. Likewise, chilling increased the incidence and severity of the disease, when it occurred during the incubation period. However, animals in which one or more limbs were traumatized failed to show any increased predisposition over controls, nor was there any correlation with the location or extent of the paralysis.[39]

Factors of Climate and Season: The view has frequently been expressed that poliomyelitis tends to be more frequent and severe in

the temperate climates, although its distribution is world-wide.[3] This view is not universally held.

However, the highly characteristic seasonal variation, with concentration in the summer months, is a universal observation. It is interesting to note that the seasonal increase in susceptibility is found in experimental animals under laboratory conditions as well as humans. It was observed, incidentally, in a recent research project that monkeys succumbed more readily and severely to inoculation when they were inoculated during the summer months.[40] This constant feature of epidemic poliomyelitis has been used as the keystone of several theories, which seek to explain the essential features of the epidemiology of this disease. The more important of these may be summarized briefly:

1. One school believes that, in the summertime, the virus is simultaneously rendered more capable of multiplying outside the human body and undergoing mutations from a benign intestinal parasite to a virulent central nervous system parasite. It is further suggested that the common gastro-intestinal complaints facilitate this process.

2. Aycock believes that the essential feature of "autoarcesis" (which he defines as pertaining "to those properties of host resistance, which cannot be explained by specific immunity due to the formation of antibodies") resides in the ability of individuals to adapt to the warmer seasons. He believes that this ability is deficient in some people and they are thereby rendered less resistant to poliomyelitis.

3. Petersen has been impressed by the meteorological changes in relation to the occurrence of poliomyelitis in epidemic proportions. More specifically, he believes that the epidemics are influenced by declining sunlight and points out the occurrence of the peaks of poliomyelitis epidemics at the autumnal equinox.[39]

These theories illustrate the widely divergent interpretations to which the known facts of the complex problem are subject.

Pregnancy: In the analysis of a relatively small series, Aycock has demonstrated statistically that clinical poliomyelitis occurred more frequently in pregnant women than in nonpregnant females of the same age group during the same epidemic.[41] Nor is this observation without confirmation. It was further observed that the incidence was significantly higher in the first trimester, when the child was

a male, and in the third trimester, when the child was a female. This latter observation is very difficult to evaluate, especially since other workers have reported increased resistance in the first trimester and increased susceptibility in the second and third trimesters.[42] It has been adequately demonstrated that pregnancy predisposes to poliomyelitis, and the role of sex hormones in susceptibility to poliomyelitis in experimental animals has been noted. Whether there is any fluctuation in susceptibility during the course of pregnancy remains to be determined by analyses of larger series.

Nutrition: In general, one should expect that poor nutrition would impair resistance to any disease. This is apparently not the case with poliomyelitis. Some investigators have attempted to demonstrate that vitamin deficiencies, notably deficiency of B, C and D, predispose to poliomyelitis. These studies were often incompletely controlled and invalidated.[3]

On the contrary it has been frequently observed that it is the healthiest and most robust children that are usually afflicted. Such observations date back to von Heine, one of the first to describe this clinical entity accurately.[43] But, robust appearance does not rule out the possibility of some specific nutritional deficiency. Carefully controlled observations on mice offer highly suggestive results. If mice are placed on a vitamin $B_1$ deficient diet they become more resistant to subsequent inoculation of the virus (Theiler and Lancing strains) than animals on an adequate $B_1$ intake. The same is true of mice whose dietary intake has been restricted to 40 per cent of their usual requirements.[44] Similarly, mice on an adequate diet have a higher mortality rate and succumb to inoculated virus more rapidly than mice on a riboflavin deficient diet.[45] It has also been observed that if inoculated mice on a vitamin $B_1$ deficient diet are given $B_1$ after they have apparently resisted the infection, they developed paralysis after a prolonged incubation period.[46]

These observations are entirely consistent with the earlier beautifully controlled studies of Howe and Bodian on virus-refractory states in neurones. They found that chromatolyzed neurones were refractory to the virus, while healthy neurones are highly susceptible. Furthermore, repeated trauma to nerve fibers rendered them incapable of transmitting the virus, even after complete

regeneration.[5] The correlation between these studies is highly suggestive in the light of the well-known importance of thiamine and riboflavin in nervous system metabolism, and the clinically known neuritis that results from their deficiency. Although these observations are highly suggestive they are not sufficiently established to warrant their application clinically in the prevention of severe central nervous system involvement of poliomyelitis.

Constitution: The so-called "poliomyelitis type" was first studied carefully by Draper in his well-known constitution clinic. Consistent with the earlier clinical impression, his statistical studies revealed that the most susceptible child is "the large, well grown individual, who has definite characteristics of face and jaw, is broad-browed and broad and round of face."[47] In general, they were characterized by "large bodies and small gonads," "overgrowth and underdevelopment." He later found that their rate of growth tends to be faster before and after puberty, in contrast to the more uniform rate of growth amongst controls.[47] All in all, they appear to be subclinical hypopituitary types. He also found statistically significant correlation between brown spots, long curved eyelashes, large central incisor teeth, etc. and susceptibility to poliomyelitis.

Aycock's constitution studies, on the other hand, reveal a statistically significant correlation with increased arm length, leg length and body length suggestive of mild hyperpituitarism.[43] These studies are little more than slightly suggestive of relative hormone imbalance in the susceptibility to poliomyelitis, and confirmation of such a belief awaits much more accurate quantitative studies.

Autoarcesis: The concept of autoarcesis was introduced by Aycock and covers the various factors in the host on which nonspecific immunity is based. He has pursued this problem along three major lines:

1. Epidemiologic and Genetic. There is fairly well substantiated evidence for hereditary predisposition to paralytic poliomyelitis. For example, in one series, 51 per cent of cases gave a family history of poliomyelitis, while only 5 per cent of unaffected individuals gave such a history. Other genetic studies have shown that, in a small series of 29 paralytic cases, poliomyelitis could be traced in both the maternal and the paternal pedigree in half the cases, while the stigma could be found in one pedigree in the other

half. Furthermore, it was found that in such families 1 out of every 5.5 individuals were afflicted. If predisposition is hereditary and carried by a recessive gene, the expected incidence would be 1 out of 4, slightly higher than that obtained.[40]

2. Anthropometric. Aycock's finding of increased susceptibility in tall, long-limbed individuals has been discussed above.

3. Experimental. Aycock has found that when immature female monkeys are castrated and then treated with estrin, some do not develop poliomyelitis on subsequent inoculation, and those that do have a longer incubation period. Whereas, similarly castrated female monkeys that were not treated with estrin, all developed paralytic poliomyelitis on inoculation.[43] This is not entirely consistent with Aycock's studies on the predisposing influence of pregnancy, but is suggestive of the influence of hormone imbalance.

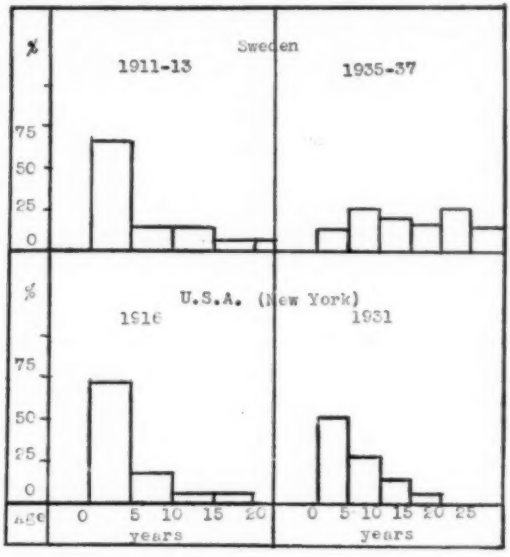
## PREVALENCE OF POLIOMYELITIS

An accurate estimate of the full extent of the prevalence of poliomyelitis is impossible to obtain due to the difficulty of diagnosis of the more mild forms. However, some idea of the number of people in whom the diagnosis is made and reported may be obtained from the number of reported cases.

Poliomyelitis occurs in sporadic and epidemic proportions in various parts of the United States every year, ranging from 136 cases in 1908 to 27,667 cases in 1916. In more recent years, the number of reported cases has varied between 2,000 and 10,000. One calculated average morbidity rate (years 1931-1937) is 6.5 cases per 100,000 population. The annual toll of lives (since 1911) taken by poliomyelitis has varied between 720 and 7,130, though most commonly it falls around the lower end of this range. In terms of mortality rate, between 0.7 and 10.0 people out of every 100,000 die from poliomyelitis each year, usually somewhat less than 1 per 100,000. In nonepidemic years, of all the diagnosed reported cases, about 13.5 per cent are fatal.[39]

Age Incidence: The morbidity and fatality figures cited above assume greater importance when it is considered that they represent chiefly the younger members of our population—individuals who have not yet reached the threshold of productive life. Poliomyelitis is primarily a disease of childhood and early adolescence, the vast majority of the cases occurring below the age of 15 years. If this age group is subdivided into infancy, childhood and early

adolescence, it will be found that most of the cases occur in the latter two groups. This is readily seen from the following fairly typical age-distribution table:[39]




(from Burnett, F. M., Med. J. Australia, p. 325, 1940, 1).

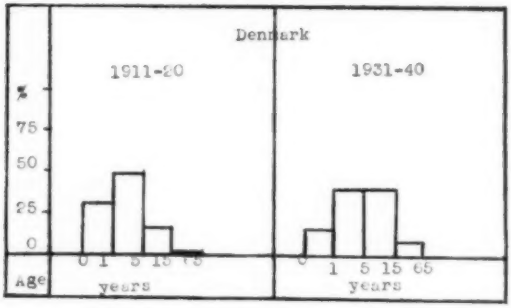



Fig. 5. Shifting Age Incidence in Poliomyelitis. (constructed from data in Bertinius[39], Acta. path. et microbiol. Scandinav., Supp. LXVIII, 1947).

| Age Group | % of Cases |
|---|---|
| 0 — 1 year | 14.4 |
| 1 — 4 years | 39.0 |
| 5 — 14 years | 39.4 |
| 15 — 64 years | 6.9 |
| 65 plus years | 0.2 |

The age distribution of poliomyelitis is apparently not static. Investigators in different countries[3,39] have observed that poliomyelitis has become more prevalent in childhood and early adolescence, and less prevalent in infancy. This shift of the age distribution curve is depicted graphically in Fig. 5.

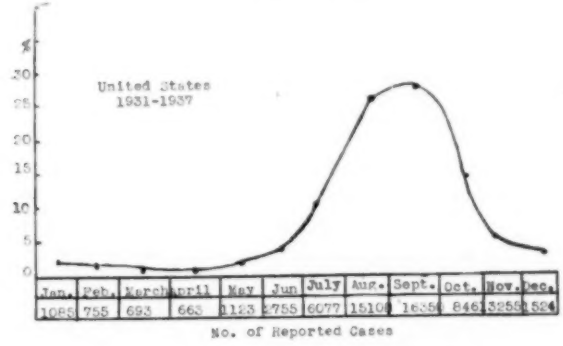



Fig. 6. Monthly Incidence of Poliomyelitis. Average monthly incidence of poliomyelitis in per cent and numbers for United States, 1931-1937 (constructed from data in Bertinius,[39] Acta path. et microbiol. Scandinav., Supp. LXVIII, 1947).

Sex: There is no significant difference in sex incidence.

Geographic Distribution: The distribution of this disease is world-wide. No part of the world where records are kept fails to reveal the occurrence of poliomyelitis in at least sporadic proportions. However, it is primarily a disease of the temperate zones. It is a commonly held opinion that the severity and frequency of the disease is approximately proportional to the distance from the equator.[3]

Seasonal Prevalence: The seasonal incidence of poliomyelitis epidemics is a constant characteristic of the epidemiology. In the United States, the epidemic usually begins in June and rises gradually to its peak in August and September. By October, the

epidemic begins to fall more rapidly than it rose and tapers off into December. Sporadic cases occur throughout the rest of the year[17] (see Fig. 6). In warmer climates the epidemic usually begins earlier.

Rural vs. Urban Prevalence: It has not infrequently been observed in this country that during an epidemic the frequency and severity of the disease tends to be higher in rural and suburban areas than in urban areas.[17] However, a recent statistical study of paralytic cases occurring over a period of years in the early 1900's in Sweden, reveals that there was no difference in the urban and rural incidence in the most populated and the most frequently attacked regions.[39] It would appear, that the rural and urban prevalence may be different in any given epidemic, but they tend to equalize over a period of years.

## METHODS OF CONTROL

The preventive measures employed at the present time are inadequate to control epidemic poliomyelitis. The best that can be hoped for is to decrease the chances of infection till the epidemic runs its course to termination in the early winter.

The principles upon which a rational control program is based are as follows:

1. The virus is widely disseminated during an epidemic.
2. The virus is present in infective doses in the throats and stools of healthy individuals.
3. The most important mode of transmission is by direct contact, contaminated hands and fomites.
4. Although insect transmission has not been established, flies are known to carry infective doses of virus during an epidemic.[50]

The preventive measures which may be employed during an epidemic may conveniently be divided into community measures and individual measures.

Community Measures: Efforts should be made to prevent intimate contact between children by closing playgrounds, swimming pools, etc. where children are apt to be herded together. Schools should be closed only if this measure serves its purpose; closing schools in a densely populated area is apt to increase contact between idle children, whereas closing schools in a rural area will serve its purpose.

Isolation: Once the diagnosis is made the patient should be

isolated for 2 to 4 weeks after the onset of symptoms. The indications for the institution and duration of isolation will be better defined when more practical methods of detecting the virus are available. The isolation is better carried out in a hospital, general or contagious. The same isolation technique that is used with typhoid should be used with poliomyelitis.

Quarantine: Although the virus is known to occur frequently in the close contacts of an active case, it is impractical and probably unneccessary to quarantine all close contacts. However, all household contacts who are food-handlers or who come into intimate contact with children should be quarantined for two weeks.

Fly Control: It is doubtless wise to prevent breeding of flies and to prevent their access to human sewage in so far as is possible without recourse to extraordinary measures. However mass-spraying with insecticides, such as DDT, is of doubtful value.

Vaccination: There is no safe effective vaccine against poliomyelitis. Many attempts have been made to prepare one, but none have been demonstrated to be beneficial. Indeed, in one series of vaccinated children there was evidence that the vaccine had caused poliomyelitis in 10 of them.

Individual Measures: Since poliomyelitis produces mild or no symptoms in most of the individuals infected, but very severe and permanent disability in those few that develop the central nervous system form, one should ideally like to exercise special precautions for those who are more susceptible to the paretic form. Unfortunately, little is definitely known about the nature of this susceptibility. However, there is fairly good evidence that hereditary predisposition to paretic poliomyelitis exists. Also, statistical studies suggest that pregnant women are more susceptible.

1. Therefore, children who give a family history of paralytic poliomyelitis and pregnant women should be removed from areas of severe epidemic in so far as is practicable. And, for these individuals, the other measures should be more rigidly enforced.

2. Avoid Exhaustion: Any attempt to subdue a child in his play will probably present a very difficult problem. However, every effort should be made to prevent fatigue and chilling in any child who has been exposed to an active case. Children with a family history of poliomyelitis should be strongly admonished against strenuous exercise during an epidemic.

3. Immediate Bed Rest: The devastating effects of paralytic poliomyelitis apparently can be aggravated by failure to get immediate bed rest in the prodromal stage. Therefore, any acute febrile illness, occurring during an epidemic, should be treated with immediate bed rest.[51]

4. Avoid Tonsillectomy and Tooth Extraction: The oral and pharyngeal mucosae are portals of entry for the virus and should be maintained as healthy as possible. Although there is some question about whether tonsillectomy significantly influences the incidence over a large number of cases, there is good evidence that it has been instrumental in producing bulbar poliomyelitis in some, however few, individuals.[52] That is to say, tonsillectomy is not a predisposing factor of importance most of the time, but, when it is, dire consequences follow.

5. There is no evidence that chemotherapy, blocking the nasal mucosa with chemicals, nose drops, gargles, etc. have any prophylactic value.[51]

6. Immune Sera: Large doses of immune sera have been of prophylactic value in animal experiments. Comparable human doses would range around 200 to 300 cc. of serum. The application of smaller doses has not been demonstrated to be of any value.[3]

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# CLINICAL-PATHOLOGICAL CONFERENCE

## WILLARD PARKER HOSPITAL, NEW YORK

*Meeting held January 21, 1949*

DR. PHILIP MOEN STIMSON, presiding.

### TUBERCULOUS MENINGITIS; PULMONARY TUBERCULOSIS

DR. MAZURSKY presented Case No. 5215, a 10-month-old white male, who was admitted here from another hospital on December 28, 1947 with a diagnosis of right pulmonary tuberculosis and tuberculous meningitis.

*History of Present Illness.* J. G., the patient, was admitted to another hospital on December 25, 1947, at 10 months of age, because of progressive lethargy, and occasional vomiting which had started a week prior to admission following a fall from his crib. The infant was said to have had clonic movements of all extremities the day before admission. Physical examination and x-ray revealed pathology in the right midlung field suggestive of tuberculous involvement. A spinal tap revealed 78 lymphocytes, Pandy 4+, sugar 10 mgm., negative smear and pellicle formation in all specimens on standing for thirty minutes. Neurological examination revealed no abnormal findings.

*On Admission* to Willard Parker Hospital the signs referable to the nervous system, such as nuchal rigidity, positive left Babinski, left internal squint, and absent abdominal reflexes, were elicited. A spinal tap on admission corroborated the previous findings. Streptomycin (125,000 units given intramuscularly every three hours and 100,000 units intraspinally given daily) was started.

*Hospital Course.* During January 1948 and the first two weeks of February 1948, the patient's condition seemed progressively worse, with much wasting and loss of weight, vomiting which at times was projectile, lethargy bordering on coma, low grade temperature, opisthotonus and a variety of rapidly changing neurological findings.

Nutrition was maintained by intravenous and gavage methods. Almost daily spinal fluid examinations, however, began to show a gradual increase in sugar within 2-3 weeks after admission from

10-15 mgm.% to 44 mgm.% on January 6, 1948 to 57 mgm.% on February 5, 1948. The spinal fluid protein, also, fell from 600 mgm.% to 400 mgm.% about this time. Cell counts changed between 150-350. From the middle of February 1948 to the end of April 1948 the patient showed marked slowly progressive improvement, became alert, very active, was able to take nourishment by bottle, became afebrile, and many of the abnormal neurological findings including nuchal rigidity, spasticity and Babinskis disappeared. Intrathecal streptomycin was discontinued on March 10, 1948 and intramuscular streptomycin on April 20, 1948. However, on March 4, 1948 it was noted that the patient was apparently deaf and blind. The latter finding was confirmed by the ophthalmologist who found extensive bilateral optic atrophy; no tubercles were noted. A spinal fluid examination on May 4, 1948 revealed a cell count of 40, sugar 61 mgm., protein 40 mgm.%. X-rays of the chest, taken at intervals since admission, showed beginning resolution of the right lung consolidation beginning about the middle of February. The child continued to be playful and cheerful. However, he also continued deaf and blind, and was behind normal in his development. There was no change in the neurological status from the end of April 1948 to the middle of June 1948, but a spinal fluid examination on June 8, 1948 showed that the sugar had fallen to 15 mgm.% and acid fast bacilli were found in the fluid. Accordingly, on June 10, 1948, streptomycin therapy was resumed, giving one-half of a gram, twice a day intramuscularly and 100 mgm. intrathecally once a day; this was continued to September 10, 1948. During this time the patient ran a temperature ranging from normal to 103° F. up until August 28, 1948 after which it never rose above normal. The physical condition of the patient remained essentially unchanged, except for the considerable clearing occurring in the right chest. During this second course of streptomycin the spinal sugar rose gradually to reach 50 mgm.% on July 8, 1948. The spinal sugar remained normal thereafter. The total protein, however, also rose from 300 mgm.% to 2,712 mgm.% on July 19, 1948 and was taken as evidence of a block. However, from this high level it fell gradually to 179 mgm.% by the time the second course of streptomycin was completed. The cell count ranged between 90 and 300.

On October 23, 1948 the patient, being afebrile and playful,

the physical examination being normal except for blindness and deafness, and the spinal fluid being normal except for a slightly increased protein, the patient was discharged to the follow-up clinic. X-ray of chest before discharge showed only residual fibrosis.

On November 16, 1948, three weeks after discharge, the child was re-admitted because of fever and vomiting. The child had done well at home until November 16, 1948 when fever, irritability, vomiting and anorexia occurred. Physical examination revealed no findings not present at discharge, except that the patient appeared to be able to see. Spinal tap on admission revealed 58 mgm.% sugar, 200 mgm.% protein and 155 cells, and negative smears. On November 22, 1948, streptomycin, 1 gm. daily and promizole 1½ gm. daily, were started. Re-examination of the fundi showed no evidence of optic atrophy and child appeared to have good vision. However, irritability increased, vomiting and the food intake decreased. On November 29, 1948, the patient had tonic and clonic convulsions and cyanosis, and became comatose. Up until this time the spinal fluid had shown only moderate pleocytosis, marked increase in protein to 700 mgm.% and normal sugar. But the specimen on November 29, 1948 showed a fall in sugar to 27 mgm.% Fifty mgm. of intrathecal streptomycin in addition to promizole and intramuscular streptomycin was started.

The patient progressed further downward, having generalized convulsions on December 2, 1948. On December 3, 1948, spinal fluid could not be obtained although the needle was in the canal, and it was felt that the child had chronic basilar meningitis with spinal block. On December 5, 1948 the patient ran a high temperature and convulsed. On December 6, 1948 the child had repeated convulsions and died suddenly.

Spinal sugars had remained low after November 29, 1948. A chest x-ray on November 19, 1948 had shown no change from the film taken at time of discharge from first hospitalization.

*Autopsy Findings by Dr. Dolgopol.* The body was that of a fairly well developed and nourished white male child. A yellowish fluid oozed from both nostrils. The right pupil was slightly irregular and wider than the left. On opening the chest, the heart appeared to be normal. In the right pleural cavity several firm old adhesions were found in the region of the right middle and upper lobes. Two calcified foci, about two mm. in diameter, were present in the

right upper lobe and in the middle lobe. Close to the medial surface a calcified focus was also found on the right side of the trachea. The left lung appeared to be normal. The liver, spleen and kidneys contained no tubercles and appeared to be normal. The skull was thin and on opening the skull a superficial nick was made accidently in the cortex and about six ounces of clear yellowish fluid escaped under some pressure. The convolutions of the brain were markedly flattened. The meninges were slightly thickened but contained no tubercles over the convexity of the brain. At the base there were many firm adhesions and some edema in the region of the cisterns. The brain was very soft and, after loss of fluid, weighed 900 grams. On section, the brain showed marked dilatation of all the ventricles and of the aqueduct. The brain substance was very edematous and markedly compressed.

*Microscopic Sections were Shown by Dr. Dolgopol.* The calcified lesions of the right lung and of the mediastinal lymph node showed complete arrest of the tuberculous process and absence of tubercle bacilli. No miliary tubercles were found in the lungs or in the abdominal organs. In the brain a number of veins along the wall of all the ventricles and the aqueduct were surrounded by heavy perivascular cuffs. In one place a small tubercle was situated close to one blood vessel. The ventricles showed some loss of the ependymal lining and the presence of tubercle-like pale cellular collections in the gaps.

The choroid plexus of the fourth ventricle contained a number of tubercles; a few tubercles were present in the meninges between the fourth ventricle and the cerebellum. In the spinal cord several wide bands of collagenous tissue and a few partly fibrosed tubercles were present in the meninges. These were the adhesions which produced the block in the spinal canal. The block of the ventricular system was produced by the tuberculous meningeal process between the roof of the 4th ventricle and the cerebellum.

### BULBAR POLIOMYELITIS

Dr. Robison presented Case No. 5313, a four-year-old white male, who was admitted here on December 5, 1948 with a history of having developed sore throat, fever, nausea and general malaise. About twenty-four hours prior to admission on December 4, 1948, he was seen that afternoon at another hospital where a cough

medicine was prescribed and the child was sent home. He vomited after taking a dose of the medicine. During the night the child became progressively worse, less alert and less responsive, developed respiratory difficulty and the mother took the child to the emergency room of another hospital where he was found to have a temperature of 104° F., was dyspneic with substernal reactions and had puddling in the nasopharynx. The case was referred to Willard Parker Hospital as a possible diphtheritic croup.

*On Admission,* physical examination revealed a temperature of 104.2° F., pulse 130, respirations 32, which was regular, short and labored with super-and substernal retractions. The child was well developed, well nourished, pale and toxic and semicomatose. The pharynx was full of a large amount of mucus which, when sucked out, gave considerable relief from respiratory distress. There was no inspiratory stridor, crow or hoarseness. There was marked left palatal weakness and a diminished gag reflex. Mild neck rigidity was found, but no back spasm or hamstring spasm. Deep tendon reflexes were equal and active bilaterally with no pathological reflexes. All the extremities moved normally. There was a definite left facial paralysis.

*Hospital Course.* The patient was suctioned and laryngoscoped. The pharynx was mildly injected but no membrane was present. The vocal cords were moving normally with no paralysis or membrane present. In the trachea there was a slight to moderate amount of greenish-yellow purulent discharge which was suctioned out. A lumbar puncture revealed clear spinal fluid, no cells, Pandy trace. Sugar 40-50 mgm. Breath sounds were normal, but there were a few inspiratory râles scattered throughout both bases.

During the day the patient showed no improvement and that afternoon a tracheotomy was considered but the general consensus of opinion was that most of the pathology causing the dyspnea and cyanosis was central, and that a tracheotomy would be of no benefit. A mixture of 95 per cent oxygen and 5 per cent carbon dioxide was administered by nasal catheter. Treatment consisted of penicillin, 50,000 units every four hours, hypodermoclysis of Ringer's lactate solution, Trendelenberg position for drainage and suctioning as necessary. In the early part of the evening the patient appeared better for a time and asked for water, but soon his temperature rose to 104° F, in spite of aspirin by enema and sponge

baths. During the night he required frequent suctioning and on two occasions the breathing became shallow and more rapid. Caffeine sodium benzoate was administered with some temporary improvement. At 5:15 A.M., when respiration almost stopped and became very irregular, the child was placed in the respirator but remained there only 5-6 minutes; he was removed because of lack of improvement due to inability to synchronize with the respirator. Following this, respiration became more irregular and the pulse was not palpable. He regurgitated about 500 cc. of dark brown material presumed to be from the stomach. An ampule of adrenal was administered without success and the child expired at 6:04 A. M.

*Autopsy Findings by Dr. Dolgopol.* The body was that of a white boy, four years old, rather slender and quite tall for his age. A slightly bloody crusting was present at the nostrils. Both pupils were in middilatation, the right wider than the left. Mesenteric lymph nodes were numerous and enlarged. The heart showed nothing significant, except a few pin-point hemorrhages. The right apex was deep purple in color, but the rest of the lung tissue was fluffy and grayish pink in color. On section the lung tissue was markedly edematous and on pressure droplets of green pus appeared on the cut surface of the right upper and lower lobes. A small wedge shaped area of collapsed lung tissue was present in the right middle lobe. The stomach contained about 100 cc., of brown blood. The gastric wall was not disintegrated as happens in many cases of poliomyelitis, but there was a slight superficial autolysis of the mucosa. It must be noted that the patient had been bleeding for sometime from slight trauma of the hard palate. The brain was very large and edematous, weighing 1400 grams. It showed flattened convolutions, but little subarachnoid edema.

*Microscopic Sections were Shown by Dr. Dolgopol.* In the spinal cord occasional minute hemorrhages were seen in the gray matter in the sacral, lumbar and thoracic cord. In the cervical region a number of anterior horn cells were severely degenerated. A few microglial nodules and many perivascular cuffs were present. There was only a minimal lymphocytic infiltration of the meninges in the depth of the anterior fissure, but none on the surface of the cord. In the medulla, severe cellular damage was present. Some cells of the nuclei of the twelfth nerve and many cells of the reticular

nuclei showed severe degeneration. Many glial nodules and a few perivascular cuffs were present in the midbrain and basal ganglia; the glial nodules were present in the midbrain. Sections from the lungs showed polymorphonuclears and small lumps of brown pigment in the lumen of some alveoli. In the other areas the lumen contained edema fluid and free red cells. There was no evidence of thrombi in the blood vessels of those areas. The rest of the lung tissue was partly air-holding. There was no obstruction of the bronchi, but some desquamation and a small number of polymorphonuclears were seen in some bronchi. Other organs showed no significant microscopic pathology.

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Rh Factor Blood Incompatibility. (Revista de Psiquiatria, Santiago, 11:51, 1946). According to Beca, 9 of 11 patients with encephalopathies due to erythroblastosis with kernicterus reported in the literature showed the Vogt syndrome, whereas 2 did not exhibit neurologic symptoms. Instances of mental defect in a child due to incompatibility of the Rh factor in his and his mother's blood, without neurologic symptoms, have also been reported. Isoimmunization of the mother to the Rh blood factor plays an important role in the production of oligophrenia with or without the neurologic sequels of the kernicterus. Rh blood incompatibility is frequently the causal factor of oligophrenia of unknown origin. There are certain cases of well verified incompatibility (the mother with Rh negative blood and the child with Rh positive blood) in which erythroblastosis with acute jaundice is followed by recovery of the patient without mental and neurologic symptoms. The fact is explained by a lack of lability on the part of the neurons to degenerate. Nervous constitutional lability accounts for erythroblastic degeneration, whereas diffuse cortical lesions and subarachnoid hemorrhages are the result of nervous lesions which produce oligophrenia. Erythroblastic lesions predominate in the gray nuclei at the base of the brain, but they diffuse also in the forms of cortical atrophy and subarachnoid hemorrhages. —*Journal A.M.A.*

# DEPARTMENT OF ABSTRACTS

SITA-LUMSDEN, E. G.: ACUTE INFECTIOUS LYMPHOCYTOSIS. (British Medical Journal, 4510:849, June 14, 1947).

The author reports a case of this disease in a seven-year-old boy, because there are not many on record and because of the importance of this condition in children, especially in the differential diagnosis of infectious mononucleosis and acute leukemia. The case presented is typical of infectious lymphocytosis, especially in the mild abdominal symptoms, the paucity of physical signs, the cervical adenitis, the leukocytosis with relative and absolute lymphocytosis due to increase in normal small lymphocytes, lasting 3-5 weeks, and the negative heterophile agglutination test. A mild eosinophilia appeared as the total count fell.

S. DAVID STERNBERG, M.D.

GROVER, VICTOR: THE CLINICAL MANIFESTATIONS OF SICKLE CELL ANEMIA. (Annals of Internal Medicine, 26:843, June 1947).

The author reviews the symptomatology and findings in 48 cases of sickle cell anemia treated in the medical wards of Kings County Hospital. Nothing new in particular has been added to the literature but many interesting points are brought forth in his series of cases. All of his patients were of the Negro race. Abdominal pain was a very frequent complaint and many times offered a difficult differential diagnosis, including renal colic, acute salpingitis, acute appendicitis and acute cholecystitis. Mental deficiency, which was not previously emphasized as a feature of the disease, was found, and three of the patients were committed to mental institutions. One of the patients had priapism which was relieved by 150 R.U. of roentgen therapy to the penis. Out of 388 cases in which roentgenograms of the heart were taken, 24 were reported to have cardiac enlargement. Esophograms in two cases showed auricular enlargement. Left auricular enlargement was quoted to be unusual. None of the autopsies revealed valvular damage or myocardial evidence of rheumatic fever. There were five cases which demonstrated evidence of a prolonged P-R interval. About 85 per cent of the roentgenograms of the skull and about 57 per cent of the roentgenograms of the long bones demonstrated changes

compatible with sickle cell anemia. Very striking in this study were the variable changes in the size of both the liver and spleen. During the same hospital stay the spleen might be reported as "not palpable," and then "increased to the iliac crest". The liver might suddenly increase in size until it was described as palpable three or four fingers breadth below the costal margin with associated abdominal pain. A. J. KRAVTIN, M.D.

BING, R. J.; VANDAM, L. D. AND GRAY, JR., F. D.: PHYSIOLOGICAL STUDIES IN CONGENITAL HEART DISEASE: RESULTS OBTAINED IN FIVE CASES OF EISENMENGER'S COMPLEX. (Bulletin of the Johns Hopkin's Hospital, 80:323, June 1947).

The authors summarize the outstanding features of the Eisenmenger complex according to current concepts. These are: 1. High ventricular septal defects. 2. An aorta which overrides both the right and left ventricles. 3. Dilated pulmonary artery. 4. Cyanosis which usually develops later in life. 5. Clubbing of fingers. 6. Hemoptysis frequently. 7. A loud systolic murmur heard over the base of the heart to the left of the sternum, 8. On fluoroscopy, a prominent pulmonary conus; marked vascular shadows and expansile pulsations in the lung fields. Five cases were studied by the catheter technique. The results indicate that Eisenmenger's complex is characterized by pulmonary hypertension, both systolic and diastolic pressure elevated in the pulmonary artery. There is increased resistance in the pumonary vascular tree, the cause of which is not known, but pulmonary endarteriolitis has been seen in similar septal defects, and a similar mechanism may be involved. The pulmonary artery flow is normal or reduced. There is a reduction in the effective pulmonary blood flow. The work of the right ventricle was found to be increased so it performed one-third to one-half of the total work of the heart. The volume of blood flow to the pulmonary capillaries exceeded the pulmonary artery flow, indicating a collateral circulation to the lung was operating. An excess oxygen consumption per litere of ventilation rose, showing that pulmonary blood flow can increase to satisfy respiratory demands, unlike the tetrology of Fallot. The authors present evidence to show that shunts operate both from right to left and left to right. The fact that the oxygen content of the pulmonary artery exceeded that of the right auricle indicated a shunt from left to right and

the reduction in the oxygen content of the peripheral blood indicated a right to left shunt. Mixing of both bloods occurs in the ventricles. SUMNER HAGLER, M.D.

SEDDON, HERBERT JOHN: THE STORY OF INFANTILE PARALYSIS IN MAURITIUS. (Medical Times, 75:150, June 1947).

During World War II one of the four big outbreaks of poliomyelitis in the British Colonies took place in Mauritius in the spring of 1945. Being an island of 38 miles by 28 miles and inhabited by a half million people, the British felt it was a good place to do some epidemiological studies in addition to caring for the stricken patients. The mission sent from Britain found 1,100 cases of poliomyelitis and in nearly 800 cases they were finally able to show how the disease had spread primarily by personal contact. A hospital, medical personnel, a boiler, a tank and many splints made by prisoners of Port Louis were gathered up and a fairly good poliomyelitis unit was set up. Today the orthopedic service of Mauritius is considered one of the best in the British Colonial Empire. VINCENT A. SPINELLI, M.D.

CANE, WALTER: SPONTANEOUS HYPERINSULINISM (HARRIS' SYNDROME)—A SURVEY OF TWENTY-EIGHT CASES. (American Journal of Digestive Diseases, 14:195, June 1947).

The author attempts in this article to establish a new entity. "Harris' Syndrome," in honor of Doctor Seale Harris of Birmingham, Alabama, who, in 1924, first described the condition, hyperinsulinism. Since he has been "hyperinsulinism conscious" he has diagnosed twenty-eight such cases. He had an incidence of 1.9 per cent from 1944 to 1946. Of his cases there were 21 males and 7 females, between the ages 26 to 48 years. The average duration of symptoms was 4½ years, and the symptoms, as a rule, came between 1½ and 5 hours following meals. They occasionally were initiated or exaggerated by physical exertion. The most common symptoms noted were abdominal pain (87 per cent of cases), weakness, numbness, cold perspiration, tremor, dizziness, absentmindedness and lack of alertness. Loss of weight was found in 29 per cent of the cases. Eighteen per cent had diarrhea during or after the attack. The abdominal pain, which was the most prominent part of the syndrome, was explained by the previous

findings of hypertonia and hypermotility of the stomach following the production of hypoglycemia in experimental animals. The gastric tonicity and motility increase as the hypoglycemia deepens until complete tetanus of the stomach is reached. Gastric secretion and acidity are also increased in hypoglycemia. Peristalsis of the duodenum and colon are accentuated which explains the diarrhea which occurred in some cases. The diagnosis was made by the glucose tolerance test in which rapid and sharp falls in blood sugar levels were seen in three hours in most cases. There were early falls in two hours. A five hour determination was necessary in only one case. All patients had symptoms upon fall in blood sugar. The author's observations favor the opinion that a sudden marked fall of the blood sugar, no matter between what levels, is the cause of the symptoms described as insulin shock. It was also observed that blood sugar levels, which gave some patients symptoms of hypoglycemia, did not give others these symptoms. None of the author's cases required pancreatic surgery and none of his cases fulfilled the eight criteria set down by Backus as suggestive of benign neoplasm of the pancreas. All of the cases responded promptly to low carbohydrate diets. A. J. KRAVTIN, M.D.

KALISKI, SIDNEY R.: MILD HYPOTHYROIDISM IN CHILDREN. (Texas State Journal of Medicine, 43: 186, July 1947).

This paper deals with the milder form of hypothyroidism which may be called borderline, masked or incipient. The basal metabolism in children under 7 or 8 is too unreliable, the cholesterol too variable and the urine creatinine excretion determination difficult unless hospitalization is available. A history of delayed development in teething, walking and talking is of suggestive value, but osseous retardation and a favorable response to thyroid extract as a therepeutic test are the most valuable. If physical and mental development are not what is to be expected, thyroid should be tried even if laboratory signs are not supportive. Some children, especially between the ages of 3-5 years, are parodoxically hyperkinetic due to a short attention span and hence are unable to maintain a continuity of interest. S. HAGLER, M.D.

LANDAU, RAPHAEL: LEPROSY TREATED WITH PENICILLIN. (New York State Journal of Medicine, 47: 1516, July 1, 1947).

Most authorities today believe that while penicillin does not exert a bactericidal or bacteriostatic action on the leprosy bacillus, it is of definite value in the complications of the disease, lepra reaction, chronic ulcers and inflammatory conditions of the eyes. The authors report a case of leprosy in an 18-year-old male who was treated at Willard Parker Hospital with penicillin for a period of six months. Some clinical improvement with healing of ulcers and disappearance of nodules was noted after four months of treatment. The improvement followed slight erythematous reaction of the skin over the nodules following each injection of penicillin. No general reactions were noted following crude penicillin administration. The histopathological structure of the persisting nodules showed no significant changes. VINCENT A. SPINELLI, M.D.

CORNELL, NELSON W.: PENICILLIN POISONING IN A CASE OF ACUTE STAPHYLOCOCCUS AUREUS HEMOLYTICUS INFECTION OF A HIP JOINT. (New York State Journal of Medicine, 47: 1509. July 1, 1947).

The authors present a case in a 17-year-old male of staphylococcus aureus hemolyticus infection of the right hip joint with bacteremia following a carbuncle of the right thigh. The patient was treated intensively with penicillin; one penicillin product caused an alarming polyneuritis which has resulted in a prolonged and probably permanent foot drop on the left side. This was explained on the basis of some antigen being present in some of the products. The part was completely immobilized and open operation was performed at one time. The importance of immobilization of infected joints in addition to penicillin therapy was stressed, for this not only tends to cut down the severity of the infection but prevents bad deformaties when ankylosis takes place. The joint was left with limited active and passive motion.

VINCENT A. SPINELLI, M.D.

GOLDSTEIN, JOSEPH, AND CALCAGNO, PHILIP L.: FAUCIAL DIPHTHERIA IN ADULTS WITH REFERENCE TO THE EARLY DIAGNOSIS. (New York State Journal of Medicine, 47: 1511, July 1, 1947).

A case of faucial diphtheria in a 33-year-old male, who was ill for 10 days previous to hospital admission, was presented. He was treated intensively with antitoxin and penicillin but on the

fourth hospital day ectopic beats were heard. On the seventh hospital day he developed an idioventricular rhythm with rates of 10 to 11 per minute. The patient became anuric on the tenth hospital day and expired. Autopsy revealed a dilated right ventricle, hypertrophied left ventricle with muscle appearing flabby and friable. Microscopic examination revealed a degenerative myocarditis, especially marked in the interventricular septum, extending to the fibers of the conductive system. There was absence of extensive acute tubular changes in the kidneys. The authors outline several points to assist in the diagnosis of a membranous lesion: A well demarcated area of hyperemia around the membrane with absence of an inflammatory reaction; an extending membrane; the membrane of diphtheria usually covers the faucial pillars with involvement of the uvula; there is edema of the soft palate with minimal redness. Other assisting points are: Toxic appearance; presence of albuminuria; rapid, soft, compressible pulse out of proportion to the fever; rhinorrhea with blood tinging; pain is a common complaint; there may be several patches; the temperature may be high, due to secondary infections; no characteristic odor may be present. VINCENT A. SPINELLI, M.D.

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STREPTOMYCIN IN TUBERCULOUS MENINGITIS. (Glasgow Medical Journal, 29: 235, July 1948). Montgomery records the pathologic and the microscopic observations on 6 children who died while under treatment with streptomycin for tuberculous meningitis. The most remarkable feature in this group of cases is the persistence of progressive meningeal lesions due to streptomycin-sensitive organisms, while in general the systemic foci of tuberculosis showed evidence of regression. It would appear that this paradoxic situation must be related to environment rather than to the micro-organism itself, that at a certain stage the local tissue reaction prevents the direct contact between the chemotherapeutic agent and the infecting organism. Accordingly, the pathology of fatal cases adds weight to the plea for earlier diagnosis so that treatment may begin before irreversible tissue changes occur.—*Journal A.M.A.*

# BOOK REVIEWS

PEDIATRIC NURSING. By Gladys S. Benz, R.N., M.A. Cloth. Price $4.00. Pp. 638. Illustrated. St. Louis: The C. V. Mosby Company, 1948.

This book, dealing with the nursing care of infants and children in the hospital, is well written and easy to read. However, there are frequent references to the historical background of medicine which seem to be too cumbersome and out of place for a book of this nature. The author is most anxious to give credit for every bit of information with the result that there are some 2,000 references taking up 100 pages of the book. These pages could have been best omitted or the space used to better advantage. The sections of the book dealing with the admission of the child to the hospital and preparation of the child for surgery are particularly well done and could be read with profit by the graduate nurse as well as the student nurse. MICHAEL A. BRESCIA, M.D.

THE 1948 YEAR BOOK OF PEDIATRICS. Edited by Henry G. Poncher, M.D. Cloth. Price $4.50. Pp. 542. Illustrated. Chicago: The Year Book Publishers, Inc., 1948.

The 1948 Year Book of Pediatrics shows a change in editors, from Abt to Poncher. However, the new editor continues in the fine tradition established by Abt and this latest Year Book represents a fine summary of a cross section of the pediatric literature. The impression gained by this reviewer is that the new editor has used his privilege as editor to a greater extent than his predecessor and to good advantage. The brief comments following an article summary are both instructive and informative. A particularly good article summary (p. 15), with excellent photographic illustrations, is the one by André-Thomas and Hanon concerning the physiology of the neonatal nervous system which appeared in the Presse Médicale, 56:229, March 31, 1948.

MICHAEL A. BRESCIA, M.D.

ENURESIS. By R. J. Batty, M.D. Cloth. Price 9/6. Pp. 103. Illustrated. London: Staples Press, Ltd. 1948.

This little volume adds nothing new to the already voluminous

literature on the subject. However, it serves to emphasize again the difficulty of the problem by its review of the many and varied treatments that have been tried to cure this troublesome condition. Although the author discusses the psychosomatic approach to the treatment of this condition not enough emphasis is placed on this phase of the treatment while enlarged tonsils and adenoids are mentioned too often to be in the proper perspective.

MICHAEL A. BRESCIA, M.D.

OBSTETRIC ANALGESIA AND ANESTHESIA. By Franklin F. Snyder, M.D. Cloth. Price $6.50. Pp. 401. Illustrated. Philadelphia: W. B. Saunders Co., 1949.

This book is divided into two sections, the first of which is entitled "Respiratory Injuries of the Child" and the second "The Treatment of Pain During Labor." In the first section the author covers adequately the physiology and pathology of the respiratory mechanism of the fetus and newborn. The chapters on respiration before birth, intrauterine pneumonia, atelectasis and asphyxia are excellent and should be widely read. In the second section the author discusses the various agents that have been used to alleviate labor pain, beginning with morphine and ending with a chapter on local anesthetics. The advantages and disadvantages of the various agents and methods are presented more or less for the reader's evaluation to judge them mainly by the newer concepts of respiration discussed by the author in the first section of the book. We are reminded by the author "that, while the relief of pain primarily concerns the mother alone, in reality, in the practice of obstetric analgesia, injury of the child commonly becomes the chief concern." I wonder if the obstetricians have concerned themselves sufficiently with the late effects on the child by whatever agent may be used to assuage the labor pains. In quoting Schreiber (1940) the author states that "a history of asphyxia at the time of birth was obtained in 70 per cent of a group of 900 children in whom brain lesions were evident."

MICHAEL A. BRESCIA, M.D.

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# Tip from Tony

Many children put up a whale of a squawk when it comes to getting a haircut—much the same as when they're sick and forced to down bitter tasting medicine. In both instances, the power of the candy appeal offers an expedient solution. • Where oral penicillin therapy is indicated, for example, many physicians are prescribing *Dulcet* Penicillin Tablets, which look and taste so much like candy that children accept them as a treat. A cinnamon-flavored, sugar base disguises the bitterness of the penicillin, making the *Dulcet* Tablet cube practical for use either as a troche or for systemic effect. And *Dulcet* Penicillin Tablets are therapeutically sound. Each tablet contains 50,000 units of crystalline penicillin G potassium, along with 0.25 Gm. of calcium carbonate as a buffer. • Many of your adult patients, who dislike ordinary tablets, will welcome this pleasant way of taking oral penicillin. On your next prescription, insure patient acceptance by specifying *Dulcet* Penicillin Potassium Buffered Tablets. All pharmacies have them—in bottles of 12. ABBOTT LABORATORIES, North Chicago, Illinois.

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